Symbol	Name	Synonyms	Organism
MYBL2	v-myb myeloblastosis viral oncogene homolog (avian)-like 2	BMYB, B-Myb, B-MYB, MGC15600, Myb-related protein B	Homo sapiens

WikiGenes	edit this page new
UniProt	P10244, A6MXH7, B2RES5
IntAct	P10244
OMIM	601415
NCBI Gene	4605
NCBI RefSeq	NP_002457
NCBI RefSeq	NM_002466
NCBI UniGene	4605

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LNA-1 transactivated an artificial promoter carrying the cell cycle transcription factor E2F DNA-**binding** sequences and also **upregulated** the cyclin E (CCNEI) promoter, but not the B-myb (MYBL2) promoter. [2000]

We have previously shown that B-Myb activity is cell cycle **regulated** and it is controlled by the antagonistic **effects** of cyclin D1 and A. [2000]

Our work identifies B-Myb as an **interacting** partner for cyclin D1 [?] and suggest that the activity of B-Myb during the cell cycle is controlled by the antagonistic **effects** of cyclin D1 [?] and A. [2000]

Concept 3
implementation
by Robert Hoffmann

Transcriptional activity of B-Myb [?] is substantially **enhanced** in S phase through modification by cyclin A [?]/cdk2, and the evidence points squarely to the major role being played by B-Myb [?] during this phase of the cell cycle. [2003]

Our data suggest that in vertebrates the trans-activating function of B-Myb [?] is **regulated** during the cell cycle and **link** Myb functions to cell cycle progression. [1997]

We show that the cell cycle regulators cyclin A [?] and cyclin E [?] **activate** B-Myb [?] by eradicating the inhibition mediated by its carboxy-terminus. [1997]

It has recently been discovered that cell-cycle gene transcription is regulated by a core complex named LINC that switches from a transcriptionally repressive complex in G(0)-G(1) with the p130 or p107 pocket proteins and E2F4 to a transcriptionally active **complex** in S-G(2) containing B-Myb. [2009]

We found that the ability of B-Myb both to **promote** Saos-2 cells into the S phase of the cell cycle and to overcome G1 arrest **mediated** by overexpression of the retinoblastoma-related p107 protein was correlated with the capacity of B-Myb to form an in vivo **complex** with p107, but was independent of its transactivation function. [2001]

PARP-1 coactivated B-Myb- and c-Myb-**mediated** transactivation of the AR promoter, and p53 antagonized the B-Myb/c-Myb-**induced** AR promoter activation. [2008]

We demonstrate that the C-terminus of B-Myb [?] can function as a repressor of transcription, that B-Myb [?] **interacts** with the repressor molecules BS69 and N-CoR [?] and that the repression function, like the transactivation domain, contributes to B-myb transformation. [2001]

Importantly, the repressor complex that Mip/LIN-9 forms with p107 [?] takes functional precedence over the transcriptional activation **linked** to the Mip/LIN-9 and B-Myb [?] interaction since expression of p107 [?] **blocks** the activation of the cyclin B [?] promoter **triggered** by B-Myb [?] and Mip/LIN-9. [2007]

We report here that cyclin A [?]-**mediated** phosphorylation of B-Myb [?] is associated with a marked increase in transactivation function in U-2 OS cells. [1997]

Cyclin A/Cdk2-**mediated** phosphorylation apparently **releases** the negative constraint and **triggers** B-Myb transactivation potential. [1999]

B-Myb [?] repressor **function** is **regulated** by cyclin A [?] phosphorylation and sequences within the C-terminal domain. [2003]

This inhibitory effect does not involve increased phosphorylation of B-Myb but seems to rely on the formation of a specific **complex** of B-Myb and cyclin D1 [?]. [2000]

We have shown previously that the activity of B-Myb is **stimulated** by cyclin A/Cdk2-dependent phosphorylation of the carboxyl-terminus of B-Myb. [2000]

These data suggest that B-Myb is a **target** for phosphorylation by cyclin-Cdk2 and that phosphorylation of B-Myb regulates its transcriptional activity. [1999]

Besides its role in the cell cycle, B-MYB has been shown to **promote** cell survival by **activating** antiapoptotic genes such as ApoJ/clusterin and BCL2. [2005]

Expression of the B-Myb transcription factor is **upregulated** during late G1 phase of the cell cycle by an E2F-dependent transcriptional mechanism. [1998]

Furthermore, the ability of B-Myb to **activate** a reporter plasmid was **enhanced** by the cotransfection of cyclin A [?], whereas mutagenesis of the 10 identified phosphorylation sites from B-Myb **blocked** the **effect** of cyclin A [?] coexpression. [1999]

top

While co-transfection of HeLa cells with a B-Myb [?] expression plasmid **activated** the transfected SP-A [?] promoter about 3-fold, co-transfection of B-myb with cyclin A [?] and cdk-2, to enhance phosphorylation of B-Myb [?], increased transcriptional activity of SP-A [?] constructs approximately 20-fold. [1999]

In this study, we show that the promoter of the fibroblast growth factor-4 (FGF-4) gene is strongly **activated** by B-Myb in HeLa cells and it can serve as a novel diagnostic tool for assessing B-Myb activity. [2002]

In contrast, B-Myb [?], c-Myb, and p53 [?] **down-regulated** the reporter gene expression in the transcriptional direction of the INTS7 [?] gene. [2008]

Cotransfection experiments with p53 expression plasmids and expression plasmids **encoding** in-frame deletion mutations in B-myb coding sequences indicate that the DNA-**binding** domain of the B-Myb protein is required for this activity. [1994]

Promoter mutagenesis studies showed that EGF [?]-**induced** activation of B-Myb [?] promoter required both E2F [?] and EGFR [?] target sites. [2006]

Mip/LIN9 is a recently described protein with growth suppressor, as well as growth promoting effects due to its ability to stabilize B-Myb [?] and **induce** genes required for S phase and mitosis. [2007]

Cyclin A [?]-Cdk2 and cyclin E-Cdk2 **complexes** each **phosphorylated** B-Myb in a cell-free system on the same sites as in intact cells. [1999]

Activation of the Rb [?] family **repressed** E2F-responsive genes and stimulated transcriptional activators, thereby mobilizing multiple signals, such as repression of B-MYB and DEK, that were independently sufficient to induce senescence. [2007]

Consistently, forced expression of both EGFR [?] and E2F1 [?] in EGFR [?]-null CHO cells greatly **enhanced** B-Myb [?] promoter activity, compared to the vector control and expression of EGFR [?] or E2F1 [?] alone. [2006]

A Lin-9 **complex** is **recruited** by B-Myb to **activate** transcription of G2 [?]/M genes in undifferentiated embryonal carcinoma cells. [2009]

Consistent with this notion, the S phase-specific cyclin A [?]/Cdk2 was found previously to **enhance** B-Myb transactivation activity in cotransfected cells. [1998]

Moreover, siRNA-mediated knockdown of MYBL2 led to reduced expression of CDC2 (which encodes CDC2), cyclin A2 (CCNA2), and topoisomerase II alpha (TOP2A), implicating these genes in the cell cycle and suggesting that they may be downstream targets of B-Myb. [2008]

B-Myb plays an essential role in cell cycle progression, and activation by E7 is likely to contribute to the mitogenic activity of the viral oncoprotein. [1994]

This study defines a novel function of B-Myb and further suggests that the p107 N-terminus provides an interaction domain for transcription factors involved in cell cycle control. [2002]

B-Myb is a transcription factor belonging to the myb family, whose activity has been associated with augmented DNA synthesis and cell cycle progression. [2000]

These data support the concept that Mip [?]/LIN-9 is required for the expression of B-Myb [?], and both proteins collaborate in the control of the cell cycle progression via the regulation of S phase and mitotic cyclins. [2007]

The transcription factor B-Myb is a cell-cycle regulated phosphoprotein involved in cell cycle progression through the transcriptional regulation of many genes. [2002]

The transcription factor B-Myb is a cell cycle-regulated phosphoprotein and a potent regulator of cell cycle progression. [1999]

Cell cycle regulation by the B-Myb [?] transcription factor. [2003]

We discuss in this review recent findings suggesting that B-Myb [?] is a multifunctional protein that has, in addition to its transcriptional properties, the ability to interact directly with other regulators of the cell cycle. [2003]

B-Myb [?] plays an important role in regulation of the cell cycle. [1997]

Overexpression of B-Myb [?] can bypass p53 [?]-mediated cell cycle arrest. [1997]

Thus, GFD8 cells stably expressing the human B-Myb protein behaved in a manner indistinguishable from those stably expressing C-Myb for both differentiation and cell cycle parameters. [1997]

Similar to E2F-1, B-Myb is a transcription factor whose expression is regulated at the G1/S border of the cell cycle. [1999]

In many aspects the B-Myb story resembles that of a fashionable transcription factor involved in cell cycle control: E2F-1. [1999]

Given the ubiquitous expression of B-Myb within different cell types, its link with the cell cycle, and augmented expression in transformed cells, studies are in progress to define the potential role of B-Myb in human cancer. [1999]

Consistent with this timing of expression, inhibition of c-Myb and B-Myb synthesis by treatment of cells with anti-sense oligonucleotides indicates that both proteins are required for transition from the G1 to S phase of the cell cycle. [1994]

Regulation of the cell cycle by B-Myb. [2001]

In this study we have examined the mechanisms by which B-Myb regulates the cell cycle. [2001]

Our experiments suggest, therefore, that B-Myb influences cell cycle progression at two distinct levels: by inhibiting p107 and by inducing transcription of specific target genes. [2001]

Several studies using antisense constructs or antisense oligonucleotides as well as overexpression experiments suggest that B-Myb plays an important role in the transition from G(1) to S phase of the cell cycle and that B-Myb expression is cell cycle regulated. [2000]

These results confirm that B-Myb is involved in cell cycle control, and that its dysregulation may contribute to increased sensitivity to a specific class of chemotherapeutic agents. [2008]

LINC dynamically associates with pocket proteins, E2F and B-MYB during the cell cycle. [2007]

During cell cycle entry, E2F4 and p130 dissociate and LINC switches to B-MYB and p107. [2007]

We have shown that the carboxyl terminus of B-Myb [?] acts as a cell-cycle sensor that regulates the transactivation function of B-Myb [?]. [1997]

RESULTS: We found that the transcriptional transactivation potential of B-Myb [?] was repressed by a regulatory domain located at the carboxyl terminus of the protein. [1997]

Moreover, using GAL4/B-Myb [?] fusions, it was found that an acidic region of B-Myb [?], which by comparison to c-Myb was expected to contain a transcription activation domain, actually had no inherent trans-activation activity and indeed appeared to trans-inhibit c-Myb. [1993]

Furthermore, studies using a series of 'domain-swapped' mutants between c-Myb and B-Myb revealed that the C-terminus of B-Myb, which is responsible for the protein's transactivation potential, blocks transcriptional cooperation with Ets-2. [2004]

Mutation of 10 of 22 putative cyclin A [?] sites, which greatly reduces the effects of cyclin A [?] on transactivation by B-Myb [?], had no effect on the ability of cyclin A [?] to alleviate B-Myb [?]-mediated repression of alpha2(V) collagen promoter activity. [2003]

Removal of just 29 C-terminal aa increased transactivation appreciably, however, maximal activity required removal of 143 amino acids (as in B-Myb + 561). [2001]

Although transactivation by B-Myb was severely dependent on hyperphosphorylation, neither inhibiting this activity by co-transfecting Cdk2DN nor augmenting it with cyclin A [?] resulted in significant effects on DNA-binding. [2001]

We showed recently that B-Myb autoregulates its own expression through promoter transactivation. [2000]

Overexpression of CDK9 did not result in suppression of p53-dependent transactivation or inhibition of the basal activity of the promoters tested so far, demonstrating that CDK9 is a B-Myb-specific repressor. [2000]

We have now investigated the transactivation properties of B-Myb in more detail. [1997]

Deletion analysis of B-Myb shows that a specific domain in the center of B-Myb, but not the DNA-binding domain is required for HSE-dependent transactivation. [1997]

An in vivo competition assay suggests that regulatory factor(s) that binds to the CR of B-Myb [?] is required for transactivation. [1995]

Transactivation assays on wild type and mutant promoter-reporter constructs demonstrated that c-Myb, but not B-Myb [?], can transactivate the human type I collagen alpha 2 chain gene promoter via the MBS-containing region. [2003]

These results indicate that B-Myb contains DNA-binding and transcriptional activation domains similar to those of c-Myb, but a regulatory mechanism of B-Myb activity is quite different from that for c-Myb. [1993]

In vertebrate cells the transactivation potential of B-Myb [?] is concealed by the C-terminal part of the protein. [1997]

Transactivation of Myb-inducible promoters by B-Myb is repressed by a regulatory domain located at the C-terminus of the protein. [1999]

These data indicate that phosphorylation by cyclin A [?]/Cdk2 is directly involved in enhancing B-Myb transactivation activity and that levels of endogenous cyclin A [?]/Cdk2 activity may contribute to cell line-specific B-Myb function. [1998]

Phosphorylation of B-Myb regulates its transactivation potential and DNA binding. [1999]

Additional analysis revealed that the effect of phosphorylation on B-Myb transactivation potential was enhanced by phosphorylation sites in its carboxyl-terminal half. [1999]

Phosphorylation and activation of B-Myb [?] by cyclin A [?]-Cdk2 [?]. [1997]

Coexpression of B-Myb [?] and cyclin A [?] relieved this repression by phosphorylation of B-Myb [?] in its carboxy-terminal region. [1997]

Recently, phosphorylation of B-Myb [?] by cyclin A [?] was shown to enhance greatly its ability to transactivate. [2003]

Potentiation of B-Myb [?] activity by phosphorylation was not simply a consequence of overcoming the negative effect of the C-terminus, however, as the truncated protein was to a lesser extent also activated by cyclin A [?]/Cdk2 [?]. [1997]

Using phosphorylation-deficient mutant forms of B-Myb, we also show that phosphorylation is essential for B-Myb activity. [2002]

Moreover, a mutant form of B-Myb, which lacks all identified phosphorylation sites and which has little activity, can function as a dominant-negative and suppress wild-type B-Myb activity. [2002]

One phosphorylation site (Ser(581)) appeared to negatively regulate DNA binding, as mutation of this site enhanced the ability of B-Myb to bind a Myb-binding sequence. [1999]

top

However, unlike the case of SW15, phosphorylation at this site did not affect the nuclear targeting of B-Myb. [1994]

Identification of cyclin A/Cdk2 phosphorylation sites in B-Myb. [1999]

We conclude that c-Myb and B-Myb may activate a common cellular program in the GFD8 cell line involved in both differentiation and cell cycle control. [1997]

B-Myb binding site signatures occur in many of the CHR-NF-Y target genes, suggesting a specific role for this triplet in the regulation of the cell cycle transcriptional program. [2005]

B-MYB, a transcription factor implicated in regulating cell cycle, apoptosis and cancer. [2005]

Among the candidates, MYBL2, whose product is the transcriptional factor B-Myb, which is involved in controlling cell-cycle progression and apoptosis, was significantly over-expressed in primary HCCs. [2008]

Other experiments have shown that removal of the B-Myb C-terminus constitutively activates both transactivation and DNA-binding activities, suggesting that autoregulation by this inhibitory domain is counteracted by phosphorylation. [2001]

We also show that deletion of the C-terminal domain of B-Myb does not affect HSE-dependent transactivation but allows the protein to activate a promoter containing Myb binding sites. [1997]

This suggests that the ability to activate Myb binding site containing promoters is repressed in the context of full length B-Myb and that HSE dependent and Myb binding site dependent transactivation are distinct functions of B-Myb. [1997]

Increasing evidence suggests that B-Myb [?] plays an important role during the late G1 and early S phases of the cell cycle. [1997]

Further experiments using a B-Myb dominant-negative protein suggested that transcriptional activation of genes regulated through Myb DNA-binding sequences was required for cell proliferation. [2001]

Because c-Myb and B-Myb have been involved in cell cycle progression, our results suggest that Tax, by repressing both c-Myb and B-Myb endogenous promoters, may bypass their requirement for cell cycle progression in HTLV-1-transformed T cells. [2000]

Like c-Myb, B-Myb also has a transcriptional activation domain containing a cluster of acidic amino acids in the region downstream of the DNA-binding domain, which consists of three tandem repeats of 51-52 amino acids. [1993]

We reviewed the transactivation potential of B-Myb [?] in yeast, taking advantage of the fact that inducible gene activation is an evolutionarily conserved process. [1997]

Overexpression of a non-phosphorylatable B-MYB mutant protects cells from UV-induced apoptosis, suggesting that its reduced phosphorylation, rather than causing its inactivation, facilitates B-MYB pro-survival activity. [2007]

B-Myb is a cell-cycle regulated transcription factor which is implicated in cell proliferation and has an essential role in early embryonic development. [2002]

These data indicate that phosphorylation of B-Myb is an essential modification for activity and that acetylation of B-Myb may play a role in B-Myb activity. [2002]

In this study we have observed that reducing B-MYB expression in primary human fibroblasts by using RNA interference results in a partial block of the cells in the G(2) phase of the cell cycle and cell death. [2005]

Mutation of one of these residues (T524) to alanine diminished the ability of B-Myb to promote transcription of a reporter gene, suggesting that phosphorylation of B-Myb at this site is important for the regulation of its activity by cyclin A/Cdk2. [1999]

Results obtained here demonstrate that the activities of B-Myb [?] and c-Myb are clearly distinct and suggest that these related proteins may have different functions in regulation of target gene expression. [1993]

Distinct changes in gene expression induced by A-Myb, B-Myb and c-Myb proteins. [2003]

We have characterized the role of c-Myb and B-Myb [?] in the regulation of human type I collagen alpha2 chain gene expression in fibroblastic cells. [2003]

Here, we discuss how B-MYB could be implicated in tumourigenesis by regulating gene expression. [2005]

Thus, these results confirm a major role for B-Myb in mediating intracellular signals controlling collagen gene expression in vascular SMCs. [1999]

CONCLUSIONS: Data indicate that B-Myb [?], which inhibits matrix gene expression in the adult vessel wall, reduces neointima formation after vascular injury. [2004]

Of the screened 1176 cancer-related genes, FOSL1, TIMP1, L1CAM, GDF15, and MYBL2 were found to be differentially expressed between the cell lines. [2009]

Enhanced B-Myb + 561 function correlated with the acquisition of DNA binding activity to a single Myb binding site (MBS) oligonucleotide as determined by bandshift assays, however, further assays showed that even wt B-Myb could bind a DNA fragment containing three MBS. [2001]

Previous studies have shown that B-Myb, a conserved member of the Myb transcription factor family, is a potent activator of the promoter of the human HSP70 gene but does not activate promoters containing Myb binding sites. [1997]

Overexpression of B-Myb in a megakaryoblastic cell line resulted in an increase in the number of cells entering S phase and, upon induction of differentiation, the fraction of cells actively endoreplicating increased. [2006]

MLE-15 cells, a cell line expressing SP-A [?] mRNA, also expressed B-Myb [?]. [1999]

We have stably transfected this cell line with constructs constitutively expressing c-Myb or B-Myb. [1997]

In summary, our data suggest that deregulated EGFR [?] signaling pathway facilitate tumor cell proliferation partly via EGFR [?] interaction with E2F1 [?] and subsequent activation of B-Myb [?] gene expression. [2006]

While A- and c-Myb behaved virtually identically in a variety of DNA-binding assays, B-Myb formed complexes of comparatively lower stability, rapidly dissociating under competitive conditions and showing less tolerance to binding site variations. [2001]

B-Myb overcomes a p107-mediated cell proliferation block by interacting with an N-terminal domain of p107. [2002]

Each B-Myb phosphorylation site contained a phosphoserine or phosphothreonine followed by a proline, suggesting that this phosphorylation is due to a proline-directed kinase. [1999]

B-Myb protein in cellular proliferation, transcription control, and cancer: latest developments. [1999]

Since its isolation exactly a decade ago, B-Myb has intrigued a growing number of scientists interested in understanding the mechanisms of cell proliferation. [1999]

This lead us to discover that the B-MYB protein half-life is increased in neuroblastoma compared to other normal or transformed human cell lines. [2007]

B-Myb is a cell-cycle-regulated member of the Myb transcription factor and, like c-Myb, has been implicated in regulation of hematopoietic cell proliferation and differentiation. [2001]

In further experiments, we show that the B-MYB protein extracted from neuroblastoma cells is hypophosphorylated. [2007]

While attempting targeting strategies for therapeutic purposes, we found that the B-MYB protein was difficult to downregulate in neuroblastoma cells using siRNA approaches. [2007]

B-MYB is more expressed in neuroblastoma patients with adverse prognostic indicators, corroborating the hypothesis that it plays an important role in this pediatric malignancy. [2007]

In contrast, neuroblastoma cells are resistant to UV-induced apoptosis and B-MYB protein levels do not change in UV-treated cells. [2007]

B-MYB is hypophosphorylated and resistant to degradation in neuroblastoma: implications for cell survival. [2007]

Thus, expression of stable, hypophosphorylated B-MYB in neuroblastoma may promote cell survival and induce aggressive tumour growth. [2007]

B-Myb [?] represses vascular smooth muscle cell collagen gene expression and inhibits neointima formation after arterial injury. [2004]

Using human cDNA probes in combination with FISH analysis, we localized MYBL2 to chromosome 20q13.1, a region that is commonly deleted in myeloid disorders. [1996]

The use of a commercially available genomic array excluded TOP2A (17q), and MYBL2, PTPT1, CSE1L, and ZNF217 (20q) as candidate genes for frequently amplified areas on these chromosomes, and contributed to refining the limits of chromosome regions involved in genomic imbalances. [2002]

The presence of the BMYB locus in rodent-human hybrids correlated with, and only with, chromosome region Xq13. [1991]

We discuss these findings in relation to the autoregulation of B-Myb by the C-terminal domain. [2001]

Physical interaction between CDK9 and B-Myb results in suppression of B-Myb gene autoregulation. [2000]

We report in this study that CDK9, the cyclin T associated kinase, which phosphorylates and activates RNA-Polymerase II, suppresses B-Myb autoregulation through direct interaction with the carboxyl-terminus of the B-Myb protein. [2000]

Thus, B-MYB is regulated by temperature to activate genes required for cell survival. [2005]

Temperature-dependent modification and activation of B-MYB: implications for cell survival. [2005]

Only human or murine fibroblasts exposed to high temperature are sensitized to cell death in the presence of dominant-negative B-MYB. [2005]

These data provide insight into the influence of B-Myb in human breast cancer, which is of potential clinical importance for determining disease risk and for guiding treatment.Oncogene advance online publication, 1 December 2008; doi:10.1038/onc.2008.430. [2008]

In vitro and in vivo analysis of B-Myb in basal-like breast cancer. [2008]

Here, we showed that B-Myb expression is a significant predictor of survival and pathological complete response to neoadjuvant chemotherapy in breast cancer patients. [2008]

The anti-MM effect of Aplidin is associated with suppression of a constellation of proliferative/antiapoptotic genes (e.g., MYC, MYBL2, BUB1, MCM2 [?], MCM4, MCM5, and survivin) and up-regulation of several potential regulators of apoptosis (including c-JUN, TRAIL, CASP8, and Smac [?]). [2008]

Although p16(INK4A) did not affect the genome-wide transcription changes mediated by SAHA, a small number of apoptotic genes, including BCLXL and B-MYB, were differentially regulated in a manner consistent with attenuated HDACi-mediated apoptosis in arrested cells. [2005]

top

The c-Myb, A-Myb and B-Myb transcription factors have nearly identical DNA-binding domains, activate the same reporter gene constructs in animal cells, but have different biological roles. [2003]

We show that B-Myb physically and functionally interacts with components of the Cdc34-SCFp45Skp2 ubiquitin pathway and propose that B-Myb degradation may be required for controlling the correct alternation of events during progression through the cell division cycle. [2000]

Down-regulation of the transactivating ability of B-Myb is independent of the kinase activity of CDK9, because a kinase deficient mutant (dn-CDK9) also represses B-myb gene autoregulation. [2000]

Analyses using an affinity resin show that multiple proteins bind to the CR of B-Myb [?] and that the CR-binding proteins in CV-1 and HeLa cells are different from those in NIH3T3 cells. [1995]

B-Myb [?] functions as a transcriptional activator in CV-1 and HeLa cells, but not in NIH3T3 cells, indicating that B-Myb [?] is a cell type-specific transcriptional activator. [1995]

We show in this report that the human myeloid leukemia cell line GFD8 is a useful model to compare the biological function of the structurally related c-Myb and B-Myb proto-oncogenes and to investigate the c-myb domains required for this function. [1997]

Mybl2 [?] (Bmyb [?]) maps to mouse chromosome 2 and human chromosome 20q 13.1. [1996]

Also found in breast tumors with high levels is B-Myb [?], a transcription factor whose expression is activated by E2F1 [?]/3 at the late G1 phase and the level is sustained through the S phase. [2006]

The B-MYB protein is quickly destroyed and apoptosis is induced in Ewing sarcoma cells exposed to UV irradiation. [2007]

(Cancer Res. 51:3821-3824, 1991) that placed the MYBL2 gene on human chromosome Xq13. [1996]

Expression levels of TFDP1 and E2F1 correlated with those of seven transcriptional targets (TYMS, DHFR, PCNA, RRM1, CCNE1, CDC2, and MYBL2) that play important roles in the G1/S transition, and down-regulation of TFDP1 inhibited growth of Hep3B cells. [2003]

These data suggest a critical role for human LIN-9, together with B-MYB, in the activation of genes that are essential for progression into mitosis. [2007]

The MYB related loci, AMYB and BMYB, were localized to specific human chromosome regions by Southern blot analysis of their segregation patterns in a panel of rodent-human hybrid DNAs using radiolabeled AMYB and BMYB probes. [1991]

B-Myb [?] is a widely expressed member of the myb oncogene family that has been shown to act as either an activator or repressor of gene transcription in a cell-type-specific fashion. [2003]

Deletion analyses of B-Myb [?] have demonstrated that the region conserved between three members of the myb gene family (CR for conserved region) is necessary for trans-activation by B-Myb [?]. [1995]

Both c-Myb and B-Myb, another member of the myb gene family, trans-activated the human c-myc promoter. [1992]

The cell-cycle-regulated Myb-family transcription factor B-Myb is crucial during S phase in many diploid cell types. [2006]

The cell cycle-regulated B-Myb transcription factor is required for early embryonic development and is implicated in regulating cell growth and differentiation. [2003]

Mutagenesis of the putative NLS's of B-Myb has identified two separate NLS's, NLS1 and NLS2. [1994]

EGF [?] stimulation and forced expression of EGFR [?] significantly increase B-Myb [?] gene activity and such increase occurs in the G1 phase. [2006]

top

Vertebrates express three different Myb family transcription factors, A-Myb, B-Myb, and c-Myb, that share a highly conserved DNA binding domain, bind to the same DNA sequences, and activate the same reporter gene constructs in transfection assays. [2003]

The demonstration that a B-Myb/LINC complex is vital for progression through mitosis in cells lacking a G(1)/S checkpoint has implications for both undifferentiated embryonal cells and for cancers in which pocket protein function is compromised. [2009]

We also identified a significant association between the G/G genotype of a nonsynonymous B-Myb germline variant (rs2070235, S427G) and an increased risk of basal-like breast cancer [OR 2.0, 95% CI (1.1-3.8)]. [2008]

OBJECTIVE: The function of B-Myb [?], a negative regulator of vascular smooth muscle cell (SMC) matrix gene transcription, was analyzed in the vasculature. [2004]

The highest frequencies for DNA sequence copy number gains were detected for SNRPN (61%); GNLY (44%); NME1 (44%); DDX15, ABCB1 (MDR), ATM, LAMA3, MYBL2, ZNF217, and TNFRSF6B (39% each); and MSH2, TERC, SERPINE1, AFM137XA11, IGF1R, and PTPN1 (33% each). [2004]

The biological significance of c-Myb, versus B-Myb, binding the cyclin B1 promoter was demonstrated by the fact that expression of inducible dominant negative c-Myb in K562 cells accelerated their exit from M phase. [2007]

We have examined the expression and function of B-Myb in megakaryocytic differentiation, during which cells progress from a diploid to a polyploid state. [2006]

Effects of B-Myb on gene transcription: phosphorylation-dependent activity ans acetylation by p300. [2002]

Using the yeast two-hybrid assay and in vivo binding assay, we investigated whether B-myb oncogene products (B-myb) can associate with each other. [1999]

Total B-Myb [?] levels were elevated in aortas of adult transgenic versus wild-type (WT) animals and varied inversely with alpha1(I) collagen mRNA expression. [2004]

However, neonatal WT and transgenic aortas displayed comparable levels of alpha1(I) collagen mRNA, likely resulting from elevated levels of cyclin A [?], which ablated repression by B-Myb [?]. [2004]

Furthermore, the MYBL2 and STK15 have been significantly overexpressed in prostate metastases, allowing a clear distinction between localized tumors and metastases. [2002]

EXPERIMENTAL DESIGN: To establish the frequency of 20q13 amplification and select the amplified cases to be studied, we used fluorescence in situ hybridization of bacterial artificial chromosome probes for three 20q13 loci (MYBL2, STK6, ZNF217) on sections of tissue microarrays containing 466 primary carcinoma samples. [2006]

Despite the technological advantage of fluorescence in-situ hybridization on tissue microarray, which allows refining regions of amplification, we were not able to recognize any of the MYBL2, ZNF217, CYP24 and STK6 genes as a particular relevant gene for melanoma tumorigenesis. [2007]

Whereas c-Myb trans-activated an SV40 early promoter containing multiple copies of an upstream c-Myb DNA-binding site (MBS-1), and similarly the human c-myc promoter, B-Myb [?] was unable to do so. [1993]

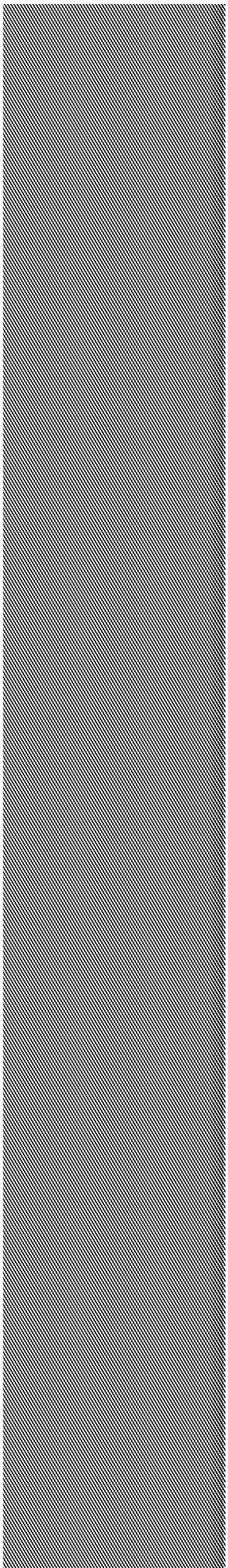
For example, in aortic smooth muscle cells B-Myb [?] represses transcription of the alpha2(V) collagen gene. [2003]

B-Myb, a highly conserved member of the Myb oncoprotein family, is a 110 kDa sequence-specific DNA binding protein expressed in virtually all proliferating cells. [2000]

Chromosomal fragmentation and other aberrations, including shorter, thicker chromatids, end-to-end fusion, and loss of a chromatid, suggest that reduced B-Myb activity is also associated with structural chromosomal instability. [2006]

The transcription factor B-Myb is essential for S-phase progression and genomic stability in diploid and polyploid megakaryocytes. [2006]

This correlates with temperature-dependent binding of endogenous B-MYB to transcriptional regulatory elements of the stress-related gene ApoJ [?]/clusterin [?]. [2005]



However, B-Myb, a more widely-expressed Myb family member, caused topo IIalpha trans-activation in both HL-60 cells and HeLa epithelial cervical carcinoma cells. [1997]

We have found that the human c-erbB-2 promoter activity is repressed by c-Myb or B-Myb in a chloramphenicol acetyltransferase co-transfection assay. [1995]

Redundant functions of B-Myb and c-Myb in differentiating myeloid cells. [1997]

The Xmyb1 cDNA clone codes for an open reading frame of 733 amino acids and exhibits a high degree of similarity over the entire predicted protein sequence with the human B-Myb protein. [1992]

Recent reports suggest a casual correlation between EGFR [?] and B-Myb [?] expression in primary breast carcinomas. [2006]

However, the roles of B-Myb in disease progression, and its mammary-specific transcriptional targets, are poorly understood. [2008]

In immortalized, human mammary epithelial cell lines, but not in basal-like tumor lines, cells ectopically expressing wild-type B-Myb or the S427G variant showed increased sensitivity to two DNA topoisomerase IIalpha inhibitors, but not to other chemotherapeutics. [2008]

This developmental transition is characterized by a switch from slow skeletal to cardiac TnI, an increase in binucleation, cardiac calsequestrin and hypophosphorylated Rb, a decrease in E2F3, B-Myb and atrial natriuretic factor, and the establishment of a more negative resting membrane potential. [2008]

To examine the role of B-Myb [?] after vascular injury, animals were subjected to femoral artery denudation, which induces SMC-rich lesion formation. [2004]

Finally, B-Myb was remarkably sensitive to cysteine-directed oxidation compared to the other Myb proteins. [2001]

Our results demonstrated that this activity was independent of B-Myb transactivation function, but correlated with its capacity to form an in vivo complex with p107. [2002]

The importance of this modification was first emphasized by showing that co-transfected dominant-negative Cdk2 (Cdk2DN) substantially reduced B-Myb transactivation activity. [2001]

Whereas wild-type B-Myb [?] transactivation activity could not be potentiated by cyclin A [?]/Cdk2 [?] in NIH3T3 cells, the truncated protein was hyperactive. [1997]

Despite this, the physical association between these proteins was not influenced by the B-Myb phosphorylation status. [2001]

We identified 10 B-Myb phosphorylation sites by automated peptide radiosequencing of tryptic phosphopeptides derived from in vivo (32)P-labeled B-Myb. [1999]

It was previously shown that B-MYB phosphorylation activates its transcriptional activity but also promotes its destruction. [2007]

B-myb proto-oncogene products interact in vivo with each other via the carboxy-terminal conserved region. [1999]

Recent studies showed that full-length B-Myb [?] containing the exon 9A **encoded** amino acids is a cell cycle **regulated** transcription factor whose activity is **stimulated** by cyclin A [?]/Cdk 2-dependent phosphorylation at the carboxyl-terminus of B-Myb [?]. [2000]

We have now determined that phosphorylation by cdk2/cyclin A **blocks** the interaction between B-Myb [?] and N-CoR [?] and that mutation of the corepressor **binding site** within B-Myb [?] bypasses the requirement for this phosphorylation event. [2002]

The B-Myb [?] transcription factor has been implicated in coordinating the expression of genes involved in cell cycle regulation. [2002]

Mybl2 [?] encodes a transcription factor that is thought to play an important role in cell cycle progression. [1996]

We have now investigated in more detail the transactivation potential of B-Myb [?] lacking exon 9A. [2000]

Our work suggests that B-Myb [?] lacking exon 9A may act as an inhibitor for full-length B-Myb [?] mediated transactivation. [2000]

Here, we show that B-Myb [?] lacking exon 9A has no transactivation activity even in the presence of cyclin A [?]. This inactivity of the shorter isoform of B-Myb [?] is not due an improper subcelluar localization. [2000]

Furthermore, by analysing the transactivation potential of Gal4/B-Myb [?] fusion proteins we have identified the amino-terminal part of the exon 9A as the principal transactivation domain of full-length B-Myb [?]. [2000]

It has been shown previously that phosphorylation of B-Myb [?] by cdk2/cyclin A enhances its transcriptional activity. [2002]

These findings provide new insights into the function of cPLA(2) in B-Myb [?]-dependent gene expression. [2004]

Binding site analysis demonstrated that both the N and C termini of cPLA(2) interact with B-Myb [?]. [2004]

Using mouse Mybl2 [?] cDNA clones as probes, we assigned Mybl2 [?] in an interspecific backcross panel to distal Chromosome 2. [1996]

In particular, NDP-alpha-MSH **down-regulated** expression of B-Myb and Myc, two oncogenes considered of paramount importance for cell proliferation and cancer. [2006]

We conclude that B-Myb [?] can **stimulate** expression of the Uchl1 [?] both in cultured cells and in vivo. [2003]

B-Myb: a key regulator of the cell cycle. [1998]

Thus these results suggest that B-Myb may be an important factor in the pathway(s) regulating collagen production in SSc fibroblasts. [2004]

Gel-shift analysis, using nuclear extracts from normal and SSc fibroblasts transfected with B-Myb, showed no differences in DNA-protein complex formation when compared with the nuclear extracts from mock-transfected cells. [2004]

Overexpression of B-Myb in SSc fibroblasts was correlated with decreased COL1A1 mRNA expression. [2004]

We investigated the role of B-Myb, a cell-cycle-regulated transcription factor, in the expression of the alpha1 (I) pro-collagen gene (COL1A1) in scleroderma fibroblasts. [2004]

Gene-expression signatures associated with loss of bmyb in zebrafish are also correlated with conserved signatures in human tumor samples, and down-regulation of the B-myb signature genes is associated with retention of p53 function. [2005]

Translection of the bcl-2-non-expressing RPMI 8226 cell line with a B-Myb expression vector induced B-Myb EMSA activity and the expression of bcl-2. [2005]

Northern-blot analysis showed an inverse relationship between COL1A1 mRNA expression and that of B-Myb during exponential cell growth and during quiescence in human SSc fibroblasts. [2004]

Transient translections localized the down-regulatory effect of B-Myb to a region containing the proximal 174 bp of the COL1A1 promoter that does not contain B-Myb consensus binding sites. [2004]

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Accordingly, the levels of B-Myb and bcl-2 proteins, and of Myb EMSA activity, were correlated over a wide range of cell lines, representing different stages of B-cell development. [2005]

B-Myb expression in vascular smooth muscle cells occurs in a cell cycle-dependent fashion and down-regulates promoter activity of type I collagen genes. [1996]

B-Myb as a critical regulator of Bcl-2 in human B cells. [2005]

Together, these results indicate that B-Myb [?] overexpression results in T and NK cell activation and increased cytotoxicity. [2001]

To assess the in vivo role of B-myb, we investigated the phenotype of mouse transgenic lines in which B-Myb [?] expression in lymphoid tissues was driven by the LCK proximal promoter. [2001]

Transgenic mice expressing a truncated, constitutively active form of B-Myb [?] in the lung epithelium showed elevated expression of UCHL1 protein. [2003]

Reporter assays indicated that the HSS8 sequence containing the three B-Myb sites may act as an enhancer when it is linked to the bcl-2 gene promoter. [2005]

Interaction of B-Myb with HSS8 may enhance bcl-2 gene expression by co-operating with positive regulatory elements (e. g. previously identified B-Myb response elements) or silencing negative response elements in the bcl-2 gene promoter. [2005]

Sequence-specific assignment of the B-Myb DNA-binding domain (B-MybR2R3) bound to a 16 base-pair DNA target site corresponding to a regulatory site from the tom-1 gene. [2003]